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CONCISE CLINICAL GUIDANCE

# 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock

A Report of the American College of Cardiology Solution Set Oversight Committee

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## PREFACE

The American College of Cardiology (ACC) has a long history of developing documents (eg, decision pathways, health policy statements, appropriate use criteria) to provide members with guidance on both clinical and nonclinical topics relevant to cardiovascular care. In most circumstances, these documents have been created to complement clinical practice guidelines and to inform clinicians about areas where evidence is new and evolving or where sufficient data is more limited. Despite this, numerous gaps persist, highlighting the need for more streamlined and efficient processes to implement best practices in patient care.

Central to the ACC's strategic plan is the generation of actionable knowledge—a concept that places emphasis on making clinical information easier to consume, share, integrate, and update. To this end, the ACC has shifted from developing isolated documents to creating integrated "solution sets." These are groups of closely related activities, policy, mobile applications, decision-support tools, and other resources necessary to transform care and/or improve heart health. Solution sets address key questions facing care teams and attempt to provide practical guidance to be applied at the point of care. They use both established and emerging methods to disseminate information for cardiovascular conditions and their related management. The success of solution sets rests firmly on their ability to have a measurable impact on the delivery of care. Because solution sets reflect current evidence and ongoing gaps in care, the associated tools will be refined over time to match changing evidence and member needs.

Concise Clinical Guidance represents a key component of solution sets. They are meant to be transitional providing guidance and application to practice prior to the evidence required for expert consensus decision pathways or clinical practice guidelines. Concise Clinical Guidance are intended to illustrate clinical decision-making processes using tools (ie, figures, tables, checklists) and are limited in scope focusing on patient populations who share certain characteristics, such as conditions, subtypes, or lines of therapy. In some cases, covered topics will be addressed in subsequent expert consensus decision pathways, appropriate use criteria, clinical practice guidelines, and other related ACC clinical policy as the evidence base evolves. In other cases, these will serve as stand-alone policy.

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*Chair, ACC Solution Set Oversight Committee*

## 1. INTRODUCTION

Cardiogenic shock (CS) is a complex, heterogenous, multifactorial syndrome in which a cardiac disorder results in insufficient cardiac output culminating in end-organ hypoperfusion.[1] CS is one of the most common causes of admission to contemporary cardiac intensive care units and remains a highly morbid and lethal complication given its dynamic and often unpredictable course, with short-term mortality ranging from 30% to 40% and 1-year mortality approaching or exceeding 50%.[2-5] Whereas CS due to acute myocardial infarction (AMI-CS) has been the most extensively studied form of CS in randomized controlled trials (RCTs), the incidence and prevalence of CS due to

nonacute myocardial infarction (AMI) causes, specifically, heart failure (HF)-related CS (HF-CS), has increased during the past decade in the United States, with notable differences in baseline characteristics, comorbidities, resource utilization, and in-hospital outcomes.[5-8] Despite advances in revascularization and increasing use of temporary mechanical circulatory support (tMCS) during the past 2 decades, RCTs have largely failed to identify treatment strategies that reliably improve mortality other than early revascularization for AMI-CS.[9] The first trial to demonstrate any benefit with tMCS was the Danish-German Cardiogenic Shock (DanGer Shock) trial, which showed that early use of a microaxial flow pump in *select* patients with ST-segment elevation myocardial infarction (STEMI)-related shock improved 180-day survival as compared with standard of care.[9a]

Recognizing the urgency of evaluating and managing CS, the American College of Cardiology (ACC) convened a virtual Heart House Roundtable of international experts, including a diverse, multidisciplinary group of stakeholders across multiple specialties, to address important unresolved issues including—but not limited to—early identification and initial evaluation and management of CS; optimal hemodynamic monitoring; pharmacological therapies; tMCS; and critical care management (see Supplemental Appendix for participant list and discussion questions). The objective of this Concise Clinical Guidance is to address pivotal questions around clinical decision making and provide actionable guidance for the interdisciplinary team involved in the evaluation and management of patients with CS.

In accordance with ACC's Relationships With Industry and Other Entities policy, relevant disclosures for the writing committee and comprehensive disclosures for external peer reviewers can be found in Appendices 1 and 2. A list of abbreviations relevant to this Concise Clinical Guidance can be found in Appendix 3. To ensure transparency, a comprehensive table of the writing committee's relationships with industry, including those not pertinent to this document, has been created. This can be found in the online Supplemental Appendix.

### 1.1. Acknowledgments

The writing committee would like to acknowledge the Critical Care Cardiology Section of the ACC for proposing CS as a Clinical Concise Guidance topic. The group would also like to acknowledge the invaluable contributions of Ashleigh Covington and Emma Spoehr for supporting the writing committee in all aspects of the manuscript development process, as well as the illustrators who helped support the visual enhancement of the figures for this manuscript.

## 2. ASSUMPTIONS AND DEFINITIONS

### 2.1. General Clinical Assumptions

1. The guidance in this Concise Clinical Guidance is intended for adult patients noting that pediatric presentations of CS differ and may require a modified Society for Cardiovascular Angiography and Intervention (SCAI) system.[10]
2. The principal focus of this document applies to patients hospitalized due to CS secondary to AMI (AMI-CS) or HF (HF-CS).
3. The guidance is intended for clinicians across a broad array of disciplines who routinely evaluate and manage patients in CS in diverse clinical settings.
4. The writing committee endorses the evidence-based approach to CS diagnosis and management recommended in the 2021 ACC/American Heart Association/SCAI Guideline for Coronary Artery Revascularization[11] and in the 2022 American Heart Association/ACC/Heart Failure Society of America Guideline for the Management of Heart Failure.[12]
5. Optimal care decisions should reflect evidence-based guidelines that incorporate the individual patient's preferences, values, and priorities, as well as those of the managing clinician and care team. The writing committee endorses a shared decision-making model framework for care delivery, especially in areas where clinical equipoise exists due to treatment uncertainty.
6. This Concise Clinical Guidance is based predominantly on expert consensus integrating the best data available and is not intended to supersede good clinical judgment, as many important clinical questions remain unanswered in CS evaluation and management. Interdisciplinary consultation, communication, and collaboration is strongly encouraged.
7. As new data emerge, they will likely inform the considerations and suggestions for clinical practice provided here. Clinicians should thoughtfully incorporate novel discoveries and scientific evidence into their clinical practice.

### 2.2. Definitions

*CS:* A cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion irrespective of underlying blood pressure.[1]

*AMI:* Defined as the irreversible necrosis of heart muscle due to myocardial ischemia. A common cause for infarction is deprivation in myocardial oxygen supply because of interruption of blood flow in ≥1 coronary arteries because of plaque rupture, erosion, fissure, or coronary dissection. The data element set for a myocardial infarction event requires both subjective and objective

findings, including symptoms, cardiac biomarkers, and electrocardiographic abnormalities. The writing committee endorses data elements that were selected based on published peer-reviewed MI definitions developed by national and international cardiovascular subspecialty societies (American Heart Association, ACC, European Society of Cardiology, and SCAI) and are commonly used by regulatory bodies that oversee the conduct of cardiovascular clinical trials.[13]

*AMI-CS:* AMI-CS includes patients with CS due to AMI in the presence or absence of ST-segment elevation on 12-lead electrocardiography (ie, STEMI and non-STEMI, respectively).[1] Consistent with the Shock Academic Research Consortium standardized definitions, left, right, or biventricular dysfunction may result from ongoing myocardial ischemia, ischemic injury, or mechanical complications of MI as the primary etiology of CS.[1] Note that CS resulting from acute bradyarrhythmias, tachyarrhythmias, or advanced heart block, postcardiac arrest, or any other complications in the setting of AMI are also classified as AMI-CS.[1]

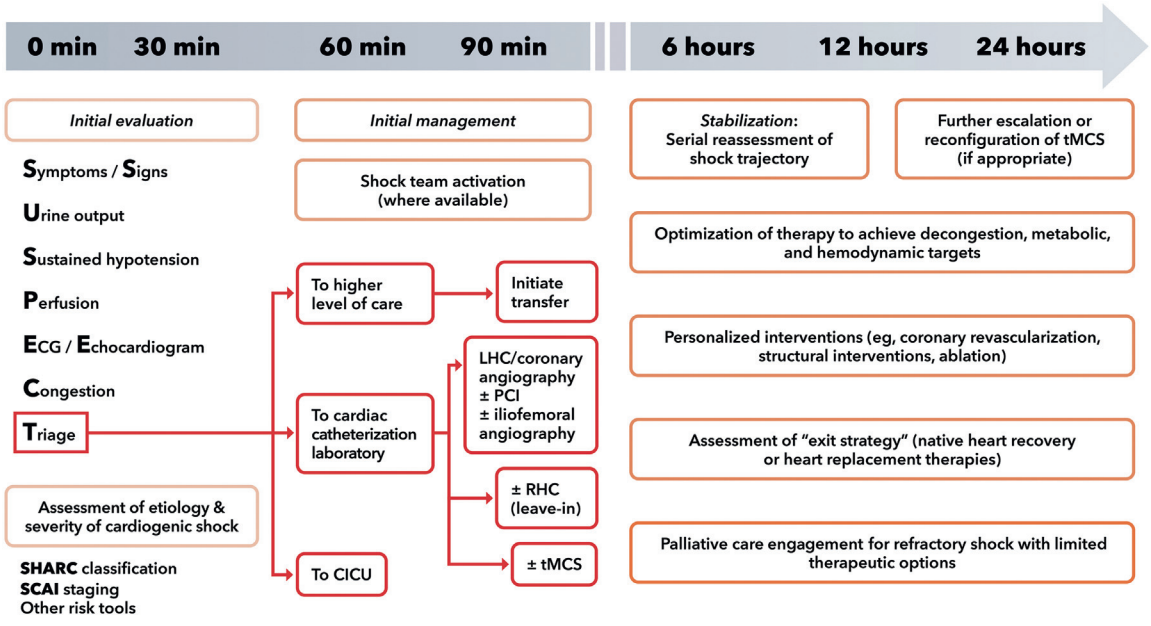
*HF:* Defined as per the universal definition of HF[14]: symptoms and/or signs of HF caused by structural/functional cardiac abnormalities *and* ≥1 of the following: 1) elevated natriuretic peptides; or 2) objective evidence of cardiogenic pulmonary or systemic congestion. A HF event, including hospitalization, is defined by the criteria outlined by the 2014 ACC/American Heart Association Key Data Elements and Definitions for Cardiovascular Endpoint Events in Clinical Trials.[15]

*HF-CS:* HF-CS is due to CS related to primary myocardial dysfunction ascribed to either ischemic or nonischemic etiologies of cardiomyopathy.[1] It may be further subcategorized into those with either *de novo* HF-CS (ie, acute myocardial dysfunction that is known or suspected to be new in onset) vs acute-on-chronic HF-CS (ie, acute decompensation of chronic or progressive HF with dilated cardiomyopathy).[1] As with AMI-CS, there may be varying degrees of ventricular involvement, including left, right, or biventricular congestive profiles.[1] HF-CS may be further categorized by the specific etiology of the underlying myocardial dysfunction, including—but not limited to—acute myocarditis, takotsubo cardiomyopathy, peripartum cardiomyopathy, tachycardia-related cardiomyopathy, hypertrophic cardiomyopathy, or infiltrative diseases such as cardiac amyloidosis or sarcoidosis, among many others.[1]

## 3. SUMMARY GRAPHIC

The summary graphic (Figure 1) represents key considerations and a proposed road map for the first 24 hours of CS evaluation and management.

**FIGURE 1** 24-Hour Roadmap in the Evaluation and Management of Cardiogenic Shock: SUSPECT CS




Sources for this figure: Waksman R, et al.[1] and Naidu SS, et al.[2] CICU = cardiac intensive care unit; CS = cardiogenic shock; ECG = electrocardiogram; LHC = left heart catheterization; PCI = percutaneous coronary intervention; RHC = right heart catheterization; SCAI = Society for Cardiovascular Angiography and Interventions; SHARC = Shock Academic Research Consortium; tMCS = temporary mechanical circulatory support.

## 4. DESCRIPTION, RATIONALE, AND IMPLICATIONS

### 4.1. Initial Evaluation of CS

Early diagnosis of CS is of critical importance, as it allows for timely intervention that may ultimately impact outcomes. Traditionally, for purposes of research and enrollment in clinical trials and registries, the criteria used to diagnose CS have included hypotension and end-organ hypoperfusion along with evidence of congestion or decreased cardiac output, (eg, measured using a pulmonary artery catheter)[3]; however, these criteria dichotomize a dynamic clinical entity across a spectrum of illness severity and they also fail to acknowledge the deleterious consequences of normotensive CS (ie, end-organ hypoperfusion without hypotension), which has been associated with increased mortality.[16,17] The diagnosis of CS, however, begins with a suspicion of an underlying state of inadequate cardiac output in patients at risk.[16] This suspicion can then be confirmed using readily obtainable clinical, laboratory, and/or imaging data. Regardless of the location of the hospitalized patient (emergency room, medical or cardiac telemetry ward, intensive care unit, etc) and across all hospitals with varying resource infrastructures, clinical symptoms, physical examination, and vital signs form the cornerstone for the initial diagnosis of CS.

Presence of hypotension (defined as systolic blood pressure <90 mm Hg, or mean arterial pressure [MAP] <60 mm Hg, or a >30 mm Hg drop from baseline for >30 minutes), a heart rate >100 beats per minute, and a narrow pulse pressure (<25% of systolic blood pressure), in isolation or in combination, should all raise suspicion for CS. Physical exam findings of lethargy, confusion, altered mental status, cold and sweaty extremities, prolonged capillary refill times (>2 s) and reduced urine output (<30 mL/h or <0.5 mL/[kg·h]), even in the absence of hypotension, should similarly raise consideration of CS.[1,18-20] A simultaneous assessment of respiratory status may yield additional signs of congestion or volume overload (eg, tachypnea, orthopnea, decreased arterial oxygen saturation, etc). More subtle symptoms of CS may include nausea, vomiting, abdominal pain, early satiety, and decreased appetite, reflecting evidence of gastrointestinal ischemia due to inadequate cardiac output. Clinical suspicion of CS should then be supplemented by readily available laboratory tests, such as a comprehensive metabolic profile assessing for acute kidney and hepatic injury; a venous or arterial blood gas with evidence of metabolic acidosis, and elevated venous or arterial lactate (>2 mmol/L). Additional laboratory data useful in the overall evaluation of patients with CS include serum sodium, and biomarkers including high sensitivity troponin and N-terminal pro brain-type natriuretic peptide.[1] A central venous catheter can be helpful to measure central venous pressure and obtain a central venous oxygen saturation.

**TABLE 1** SUSPECT CS: A Mnemonic to Aid in Confirming a Diagnosis of CS

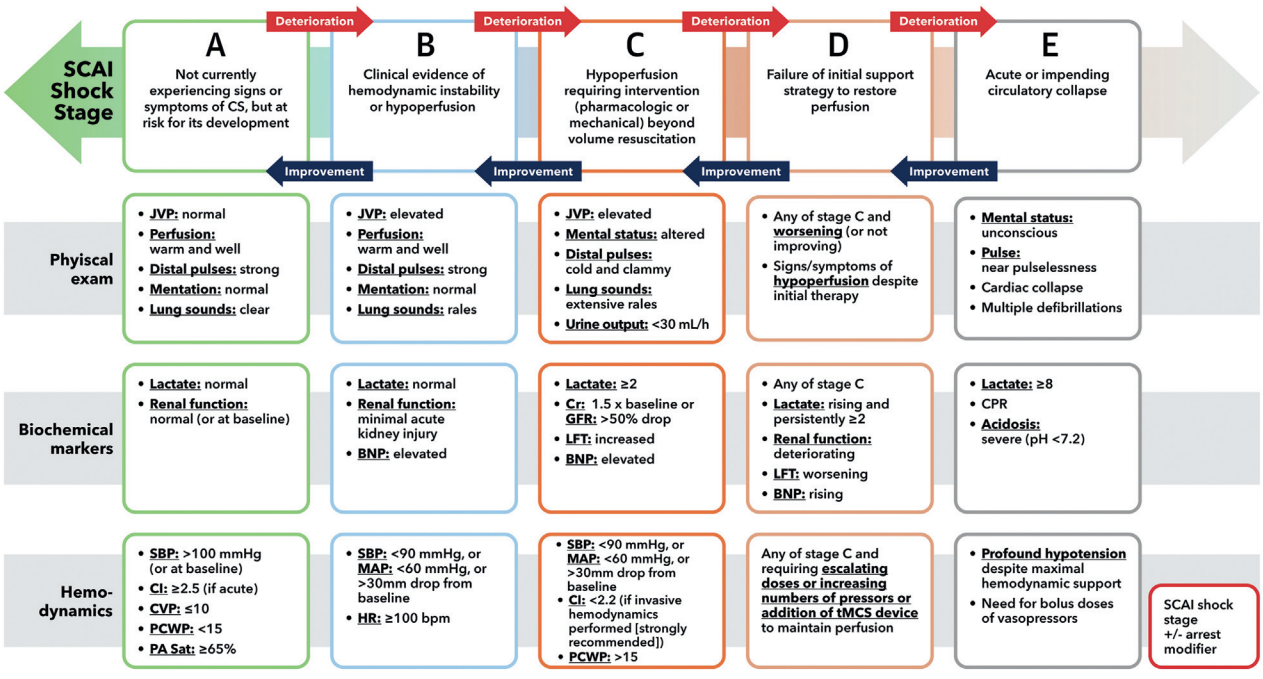
| | |
|---|---|
| **S**ymptoms/**S**igns | Altered mental status, confusion, chest pain or pressure, cold and clammy extremities, rapid pulse, low pulse pressure (<25% of SBP), elevated jugular venous pressure, crackles, rales, orthopnea, paroxysmal nocturnal dyspnea, lower extremity edema |
| **U**rine output | Oliguria or anuria, <30 mL/h (<0.5 mL/[kg·h]) |
| **S**ustained hypotension | SBP <90 mm Hg, MAP <65 mm Hg for >30 min or a >30-mm Hg decrease from baseline, or the need for pharmacological or mechanical support to maintain SBP >90 mm Hg |
| **P**erfusion | Evaluate markers of end-organ malperfusion, including lactic acid >2 mmol/L, ALT >200 U/L or >3× upper limit of normal, creatinine ≥2× upper limit of normal, pH <7.2, metabolic acidosis without another known cause |
| **E**CG/**E**chocardiogram | Evaluate acute ischemia, including ECG and sonographic evidence of STEMI (regional wall motion abnormalities); evidence of LV or RV dilation and systolic dysfunction; valvular pathology |
| **C**ongestion | Presence or absence of congestion based on physical signs and hemodynamics; elucidation of ventricular involvement (LV vs RV vs BiV) |
| **T**riage | Appropriate triage/shock team activation or possible transfer to a higher level of care |

ALT = alanine transaminase; BiV = biventricular; CS = cardiogenic shock; ECG = electrocardiogram; LV = left ventricular; MAP = mean arterial pressure; pH = potential of hydrogen; RV = right ventricular; SBP = systolic blood pressure; STEMI = ST-segment elevation myocardial infarction.

Every patient suspected to be in CS should have a 12-lead electrocardiogram and, where available, a transthoracic echocardiogram or point-of-care cardiac ultrasound[1] performed by an experienced clinician. Electrocardiographic evidence of acute ischemia, particularly ST-segment elevation, should lead to early triage to the cardiac catheterization laboratory, either locally or at the nearest capable facility, and revascularization as appropriate to coronary anatomy.[21] Sonographic evidence of diminished right ventricular (RV) or left ventricular (LV) systolic function, cardiac tamponade, or acute valvulopathies should prompt a timely consult to a general or interventional cardiologist, cardiac surgeon, or an advanced HF cardiologist, as appropriate.[22] To aid clinicians in confirming a suspicion of CS, the writing committee proposes the mnemonic "SUSPECT CS" (Table 1), the components of which include the minimum necessary criteria needed to make an early diagnosis of CS.

It is important to highlight that the initial suspicion and diagnosis of CS does not require invasive hemodynamics[1]; however, invasive hemodynamics are often useful in elucidating the ventricular involvement and congestive profile of the CS patient and may inform therapeutic decision making. Once a diagnosis is made, the severity of

**FIGURE 2** **CS Working Group-SCAI Shock Criteria for Classification of CS Severity**


SCAI Shock Stage
A
Not currently experiencing signs or symptoms of CS, but at risk for its development
B
Clinical evidence of hemodynamic instability or hypoperfusion
C
Hypoperfusion requiring intervention (pharmacologic or mechanical) beyond volume resuscitation
D
Failure of initial support strategy to restore perfusion
E
Acute or impending circulatory collapse
Deterioration
Improvement
Physical exam
• JVP: normal
• Perfusion: warm and well
• Distal pulses: strong
• Mentation: normal
• Lung sounds: clear
• JVP: elevated
• Perfusion: warm and well
• Distal pulses: strong
• Mentation: normal
• Lung sounds: rales
• JVP: elevated
• Mental status: altered
• Distal pulses: cold and clammy
• Lung sounds: extensive rales
• Urine output: <30 mL/h
• Any of stage C and worsening (or not improving)
• Signs/symptoms of hypoperfusion despite initial therapy
• Mental status: unconscious
• Pulse: near pulselessness
• Cardiac collapse
• Multiple defibrillations
Biochemical markers
• Lactate: normal
• Renal function: normal (or at baseline)
• Lactate: normal
• Renal function: minimal acute kidney injury
• BNP: elevated
• Lactate: ≥2
• Cr: 1.5 x baseline or GFR: >50% drop
• LFT: increased
• BNP: elevated
• Any of stage C
• Lactate: rising and persistently ≥2
• Renal function: deteriorating
• LFT: worsening
• BNP: rising
• Lactate: ≥8
• CPR
• Acidosis: severe (pH <7.2)
Hemo-dynamics
• SBP: >100 mmHg (or at baseline)
• CI: ≥2.5 (if acute)
• CVP: ≤10
• PCWP: <15
• PA Sat: ≥65%
• SBP: <90 mmHg, or MAP: <60 mmHg, or >30mm drop from baseline
• HR: ≥100 bpm
• SBP: <90 mmHg, or MAP: <60 mmHg, or >30mm drop from baseline
• CI: <2.2 (if invasive hemodynamics performed [strongly recommended])
• PCWP: >15
Any of stage C and requiring escalating doses or increasing numbers of pressors or addition of tMCS device to maintain perfusion
• Profound hypotension despite maximal hemodynamic support
• Need for bolus doses of vasopressors
SCAI shock stage +/- arrest modifier

Adapted with permission from Kapur NK, et al.[3] Source for this figure adaptation: Naidu SS, et al.[2] BNP = brain natriuretic peptide; CI = cardiac index; CPR = cardiopulmonary resuscitation; Cr = creatinine clearance; CS = cardiogenic shock; CVP = central venous pressure; GFR = glomerular filtration rate; HR = heart rate; JVP = jugular venous pressure; LFT = liver function test; MAP = mean arterial pressure; PA = pulmonary artery; PCWP = pulmonary capillary wedge pressure; pH = potential of hydrogen; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions; tMCS = temporary mechanical circulatory support.

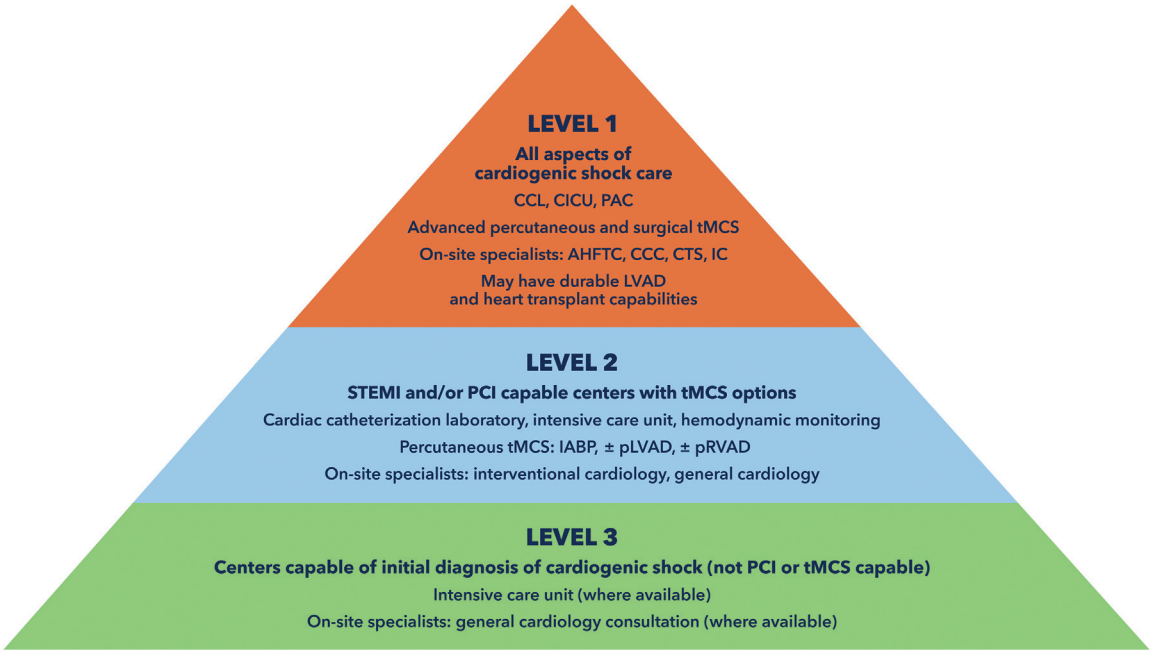
CS should be classified using the SCAI stage of CS.[2,23] An adaptation of the SCAI stage classification is presented in Figure 2.[2,3]

In addition to etiology, factors such as setting, timing, and other considerations outlined in the Shock Academic Research Consortium's Standardized Definitions for Cardiogenic Shock Research and Mechanical Circulatory Support Devices may be used to further classify the phenotype of CS, which distinguishes between the following etiologies: AMI-CS; HF-CS; postcardiotomy CS; and secondary CS (due to arrhythmias, valvular disease, pericardial disease, or other etiologies).[1] Notably, AMI-CS can be further subcategorized by whether STEMI vs non-STEMI is present; moreover, HF-CS can be subclassified by *de novo* HF-CS vs acute-on-chronic HF-CS. Once the diagnosis and severity of CS have been established, invasive monitoring or diagnostic procedures such as an arterial line and a pulmonary artery catheter may then be used to continuously monitor hemodynamics to aid in selection of and response to treatment strategies to address congestion and perfusion.[24,25] Depending upon the available resources at the hospital where the initial diagnosis of CS is made, transfer to a higher level of care (including a different institution) may be necessary for further management after initial stabilization.

### 4.2. Transferring a Patient in CS

The acuity and complexity of CS patients require an interdisciplinary, collaborative, and standardized team-based approach to management.[20,21,26] Whereas most hospitals in the United States can provide acute cardiovascular care, some can serve as CS centers (known as Level 1 CS hospital centers) being equipped with the full gamut of on-site 24/7 medical and surgical expertise, tMCS devices and "high" procedural volumes. In many cases, such CS centers may also provide advanced HF therapies such as durable LV assist device (LVAD) and orthotopic heart transplant. Advanced CS centers are typically concentrated in larger, urban cities, whereas most CS patients present to local, community hospitals

**FIGURE 3** Proposed Classification of CS Centers of Care Based on Locally Available Resources and Expertise


LEVEL 1
All aspects of cardiogenic shock care
CCL, CICU, PAC
Advanced percutaneous and surgical tMCS
On-site specialists: AHFTC, CCC, CTS, IC
May have durable LVAD and heart transplant capabilities
LEVEL 2
STEMI and/or PCI capable centers with tMCS options
Cardiac catheterization laboratory, intensive care unit, hemodynamic monitoring
Percutaneous tMCS: IABP, ± pLVAD, ± pRVAD
On-site specialists: interventional cardiology, general cardiology
LEVEL 3
Centers capable of initial diagnosis of cardiogenic shock (not PCI or tMCS capable)
Intensive care unit (where available)
On-site specialists: general cardiology consultation (where available)

AHFTC = advanced heart failure and transplant cardiology; CCC = critical care cardiology; CCL = cardiac catheterization laboratory; CICU = cardiac intensive care unit; CTS = cardiothoracic surgery; IABP = intra-aortic balloon pump; IC = interventional cardiology; LVAD = left ventricular assist device; PAC = pulmonary artery catheter; PCI = percutaneous coronary intervention; pLVAD = percutaneous left ventricular assist device; pRVAD = percutaneous right ventricular assist device; STEMI = ST-segment elevation myocardial infarction; tMCS = temporary mechanical circulatory support.

(known as Level 2 or Level 3 CS hospital centers) that may not have advanced tMCS, durable LVAD, or transplant capabilities.[22] Professional society guidelines recommend (Class 2b) transfer of patients in need of higher acuity care, beyond the scope of capabilities available at the presenting hospital, to Level 1 CS hospital centers[12]; however, at present, there is no consensus around classification of hospital centers into levels or tiers of CS care akin to trauma systems of care. Professional cardiology societies have proposed a few different approaches based on available expertise and resource infrastructure[19,20,22,27-29] but these frameworks are largely based on expert consensus (**Figure 3**). Data pertaining to the outcomes of transferred patients are scarce, pointing to an evidence gap in CS care.[30,31]

Observational studies of patients transferred to dedicated CS centers have shown mixed results with respect to associated mortality for the transferred cohort. A multicenter, retrospective observational analysis showed higher associated mortality among transferred patients that was driven by HF-related CS.[31] In a separate study comparing AMI-CS and HF-CS patients presenting initially to Level 2 or 3 CS hospital centers or a Level 1 CS hospital center in a dedicated, regionalized network across multiple hospital systems encompassing 3 states, the authors found comparable short-term outcomes (including in-hospital and 30-day mortality) and similar rates of bleeding, vascular, and stroke complications.[30] The predictors of outcomes among patients transferred for management of CS vary based on etiology of CS. Among HF-CS patients, older age, mechanical ventilation, renal replacement therapy, and multiple vasoactive drugs were associated with worse outcomes while use of a pulmonary artery catheter was associated with favorable outcomes. On the other hand, among AMI-CS patients, overweight size (body mass index >28 kg/m$^2$), worsening renal failure, lactate >3 mg/dL, and increasing number of vasoactive agents were associated with higher mortality

rates, while use of any tMCS device was associated with beneficial outcomes.[31] It is important to recognize the limitations inherent with these observational data and the merits and challenges of rigorously studying regionalized networks and systems of care in CS as an area of fertile investigation.[32]

The writing committee recommends that Level 2 and 3 CS hospital centers identify: 1) locally available resources; and 2) a dedicated CS regional center that would accept appropriately selected CS patients for further evaluation and treatment as well as might provide remote consultation for patients who may not be appropriate for transfer and/or need further stabilization before a transfer can be initiated. Conversely, Level 1 CS hospital centers should welcome consultations from their regional referring hospitals and accept appropriately selected CS patients for transfer and may provide remote consultation on CS patients who may have been deemed unstable or inappropriate for transfer. Additionally, referring hospitals are encouraged to identify on-site "CS champions" who could help facilitate CS care both locally and in concert with the CS center. Patients with AMI-CS who are triaged to a local cardiac catheterization laboratory and remain in refractory CS postrevascularization should almost always be transferred to a Level 1 CS hospital center.[22] Patients who have experienced a cardiac arrest with tenuous neurological status, or patients who have been initiated on ≥1 vasoactive medication, or those in whom a tMCS device is being considered, should also prompt communication with the CS regional center to determine whether, and when, transfer should be pursued. Once the initial suspicion of CS has been confirmed, the writing committee believes it is never too early to contact the CS regional center to discuss potential transfer vs continued management at the initial institution.[33]

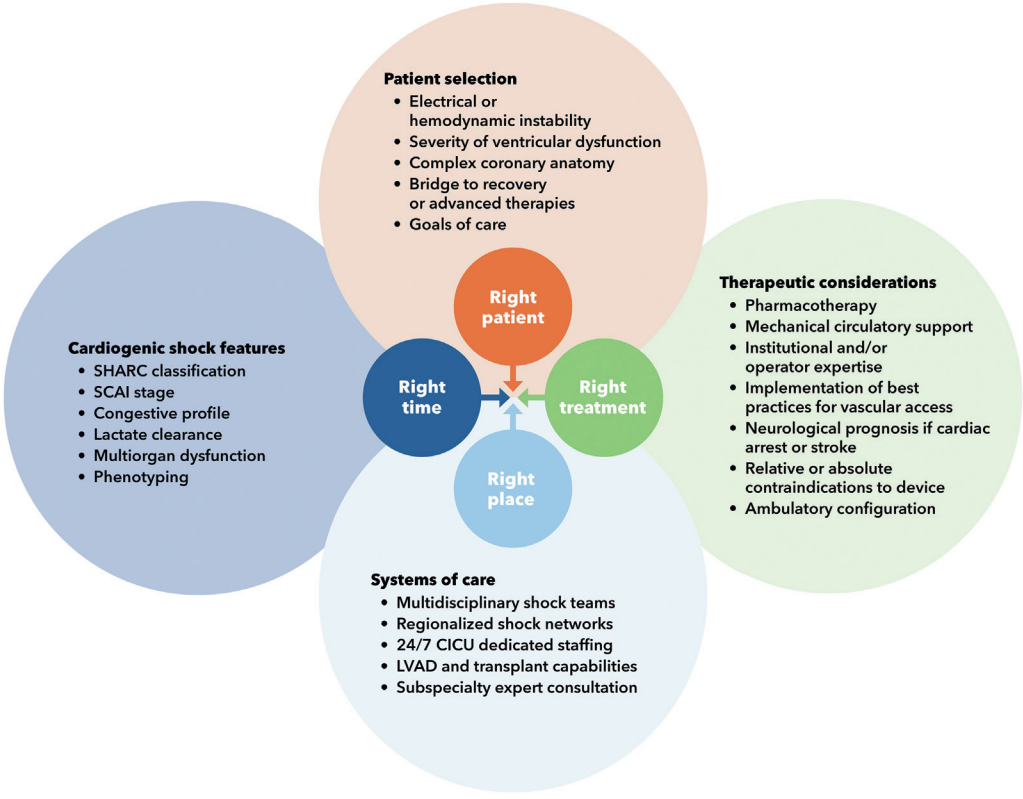
### 4.3. CS Team Activation

A standardized, interdisciplinary, team-based approach to CS management has been associated with improved clinical outcomes. Several single-center protocols initially established the proof of concept with associated lower in-hospital mortality.[34,35] A multicenter observational analysis among 10 of 24 centers with shock teams showed that shock team centers were more likely to obtain invasive hemodynamics, use advanced types of tMCS (ie, beyond an intra-aortic balloon pump), and have lower risk-adjusted cardiac intensive care unit mortality.[36] While the exact composition of CS teams has varied across institutions, key stakeholders from the following specialties have typically been included: critical care cardiology (whenever available) or general critical care in collaboration with a cardiac intensive care unit cardiologist (when it is not); advanced HF and transplant cardiology; interventional cardiology; and cardiac surgery. Extracorporeal membrane oxygenation intensivists or perfusionist and/or palliative care specialists may be included in some centers.[33] For Level 2 and 3 CS hospital centers that may not have on-site expertise or an established CS team, the writing committee strongly recommends *early* contact with the regional Level 1 CS hospital center after confirming the initial diagnosis of CS. The following key considerations may be helpful for triage, potential transfer, appropriate risk stratification, and treatment of CS patients: 1) What is the SCAI Stage and Shock Academic Research Consortium classification? 2) Does the patient need or require escalation of tMCS support at this time? 3) Are there any absolute contraindications to escalation of treatment (eg, do not resuscitate order, or terminal illness, etc)? 4) Does the institution have the resources to support this patient's anticipated needs (eg, intensive care unit bed availability, clinical expertise, and tMCS resources and availability)? 5) Is the patient hemodynamically stable for transfer?

Common elements across shock team models at Level 1 CS hospital centers include early interdisciplinary engagement and consultation to deliver necessary multifaceted care, a coordinating physician (eg, a cardiac intensive care unit cardiologist, advanced HF and transplant cardiologist, or interventional cardiologist, depending on clinical setting) for efficient patient triage, a rapid system for concurrent team activation, efficient virtual and/or bedside communication, and use of invasive hemodynamics to guide therapy selection (**Figure 4**).[21,26]

Depending on the institutional resources and personnel, all elements of the shock team model may not be readily available.[37] Additional expertise may be needed in certain clinical scenarios, such as cardiac electrophysiology for patients with electrical storm, maternal fetal medicine for critically ill pregnant patients, and adult congenital heart disease consultation for patients with complex adult congenital heart disease physiology. The writing committee affirms the importance and value of building and growing shock teams across time and frequently reviewing CS cases as patient safety and quality improvement initiatives to assess opportunities to refine protocols and strengthen relationships.

**FIGURE 4** Factors Influencing the Selection of the Right Therapy for the Right Patient at the Right Place and the Right Time



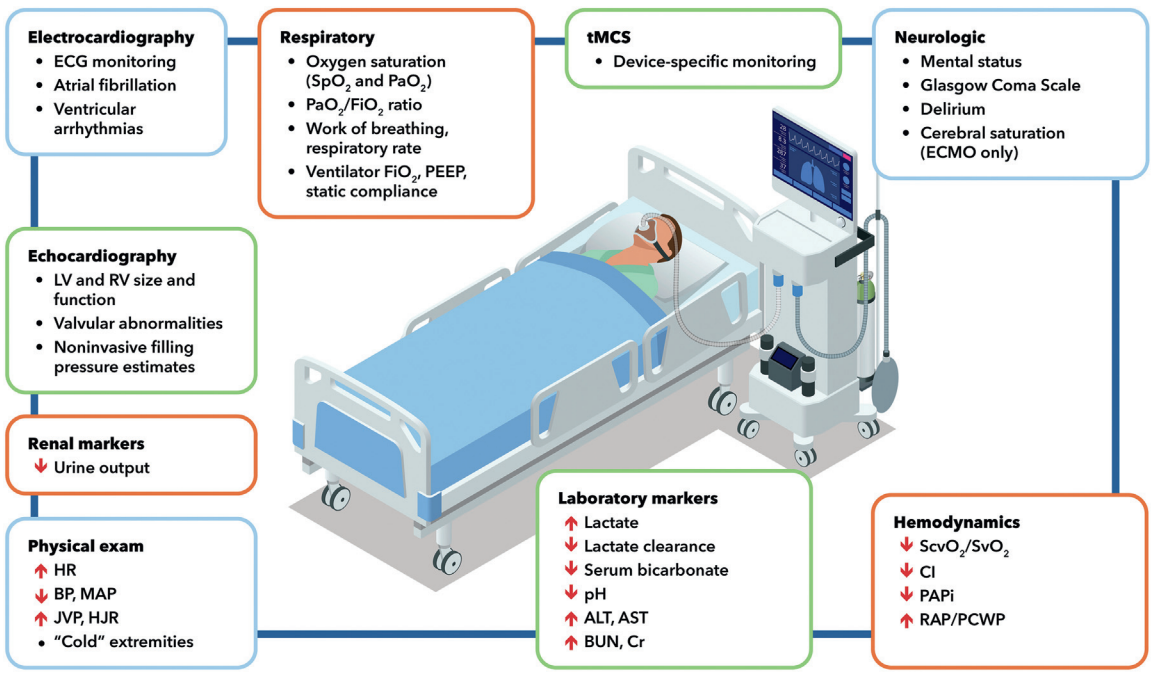

Adapted with permission from Mehta A, et al.[26] CICU = cardiac intensive care unit; LVAD = left ventricular assist device; SCAI = Society for Cardiovascular Angiography and Interventions; SHARC = Shock Academic Research Consortium.

### 4.4. Invasive Hemodynamics

Observational data suggest there is utility in applying invasive hemodynamics to characterize the phenotype of CS, assess the severity of shock, and to guide tMCS-related escalation and weaning decisions in the cardiac intensive care unit.[38,39] While there are no definitive randomized data to support the use of pulmonary artery catheters in the setting of CS, the results of the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial, studied in severe symptomatic and recurrent HF patients without CS, do not apply to the contemporary CS patient population. Both retrospective claims-based and multicenter, registry-based data suggest that the use of pulmonary artery catheters is associated with improved outcomes.[24,25,40-43] In fact, complete hemodynamic profiling as compared with incomplete or no hemodynamic assessment has been associated with lower in-hospital mortality and early hemodynamic assessment within the first 12 hours has been associated with improved clinical outcomes.[24,25] This scientific hypothesis that early invasive hemodynamic assessment (within 6 hours of randomization) decreases mortality is currently being tested in a multicenter, randomized, parallel group, adaptive trial, the Pulmonary Artery Catheter in Cardiogenic Shock (PACCS) trial, in HF-CS patients.[44]

Hemodynamics enable the bedside clinician to elucidate the congestion profile (LV dominant, RV dominant, or biventricular shock), which have been associated with adverse outcomes in both AMI-CS and HF-CS. LV dominant congestion in CS is often characterized by an elevated pulmonary capillary wedge pressure or LV end-diastolic pressure >15 mm Hg. In contrast, RV dominant congestion in CS is accompanied by a relatively normal pulmonary capillary wedge pressure in the setting of an

**FIGURE 5** Monitoring of the CS Patient in the Intensive Care Unit




ALT = alanine aminotransferase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CI = cardiac index; CPO = cardiac power output [mean arterial pressure x cardiac output]/451; Cr = creatinine; CS = cardiogenic shock; ECG = electrocardiography; ECMO = extracorporeal membrane oxygenation; $FiO_2$ = fraction of inspired oxygen; HJR = hepatojugular reflux; HR = heart rate; JVP = jugular venous pressure; LV = left ventricular; MAP = mean atrial pressure; $PaO_2$ = partial pressure of oxygen; PAPi = pulmonary artery pulsatility index [systolic pulmonary arterial pressure—diastolic pulmonary arterial pressure]/right atrial pressure; PCWP = pulmonary capillary wedge pressure; PEEP = positive end-expiratory pressure; pH = potential of hydrogen; PVC = premature ventricular contraction; RAP = right atrial pressure; RV = right ventricular; SBP = systolic blood pressure; $ScvO_2$ = central venous oxygen saturation; $SpO_2$ = oxygen saturation; $SvO_2$ = mixed venous oxygen saturation; tMCS = temporary mechanical circulatory support.

elevated right atrial pressure (right atrial or central venous pressure >15 mm Hg). Biventricular congestive profiles suggest elevation in both right atrial and pulmonary capillary wedge pressures. Both biventricular and RV congestion profiles are common in CS patients and have been associated with adverse outcomes, including death, and the need for durable LVAD and heart transplant in multicenter, observational registries.[7,42,45]

Hemodynamic parameters associated with adverse mortality in CS include low MAP, elevated right atrial pressure, an elevated right atrial pressure to pulmonary capillary wedge pressure ratio (>0.6 mm Hg), and a reduced pulmonary artery pulsatility index (the ratio of the difference between the pulmonary artery systolic pressure and diastolic pressure divided by the central venous pressure).[42,45,46] The PAPi is a derived value that reflects an amalgam of factors, including RV contractility, RV pulsatile load, and RV congestion, and is most useful in the setting of an elevated right atrial pressure >10 mm Hg and in the absence of moderate or severe pulmonary hypertension (pulmonary artery systolic pressure >50 mm Hg). Criteria for low PAPi values differ between AMI-CS (<0.9 mm Hg) and HF patients undergoing LVAD implantation (<1.85 mm Hg).[47-49] The writing committee suggests integrating invasive hemodynamic assessment with noninvasive cardiac imaging (ie, echocardiography or point-of-care ultrasound whenever available) to more precisely characterize the phenotype of CS patients (Figure 5).[27,50]

### 4.5. Pharmacological Management of CS

In CS, the goal of pharmacological therapies is to mitigate congestion (whenever present), optimize cardiac output,

**TABLE 2** Vasoactive Agents Used in CS

| Category | Agent(s) | Mechanism of Action/Receptor Binding | Dosing | Hemodynamic Effects | | | |
|---|---|---|---|---|---|---|---|
| | | | | SVR | BP | CO | HR |
| Inopressor | Norepinephrine | α1 (+++), β1 (++), β2 (+) | 0.05 -1 mcg/kg/min | ↑↑ | ↑↑ | ↑ | ↑ |
| | Epinephrine | β1 (+++), α1 (++), β2 (++) | 0.01-0.5 mcg/kg/min | ↑↑ | ↑↑ | ↑↑ | ↑↑ |
| | Dopamine | D1 (+++), β1 (++), α1 (+) | Low: 2-5 mcg/kg/min<br>Intermediate: 5-10 mcg/kg/min<br>High: 10-20 mcg/kg/min | ↑↑ | ↑↑ | ↑ | ↑↑ |
| Inodilator | Dobutamine | β1 (+++), β2 (++) | 2-10 mcg/kg/min | ↓↔ | ↓↔ | ↑↑ | ↑ |
| | Milrinone | PDE-3 inhibitor | 0.125-0.5 mcg/kg/min | ↓↓ | ↓↓ | ↑↑ | ↔↑ |
| Vasopressor | Phenylephrine | α1 (+++) | 0.1-10 mcg/kg/min | ↑ | ↑↑ | ↔↓ | ↔↓ |
| | Vasopressin | Vasopressin receptor | 0.01-0.04 U/min | ↑↑ | ↑↑ | ↔↓ | ↔↓ |
| Vasodilator | Nitroprusside | NO production | 0.3 - 10 μg/kg/min | ↓ | ↓ | ↑↔ | ↑↔ |
| | Nitroglycerin | Converts to NO | 25-200 μg/min | ↓ | ↓ | ↑↔ | ↑↔ |
| Chronotrope | Isoproterenol | β1 (+++), β2 (+++) | 2 -20 μg/min | ↓ | ↔ | ↑ | ↑↑ |
| | Dopamine | See above | | | | | |
| Inotrope | Levosimendan* | Binds to troponin C, making it more sensitive to calcium thereby improving interaction between troponin C and I | 0.05 - 0.2 mcg/kg/min | ↓ | ↓ | ↑ | ↔ |

*Not FDA approved for clinical use in the United States.

↑ = increased effects; ↓ = decreased effects; ↔ = neutral effects; (+++) = strong binding; (++) = moderate binding; (+) = weak binding; α1 = α-1 receptor; β1 = β-1 receptor; β2 = β-2 receptor; BP = blood pressure; CO = cardiac output; CS = cardiogenic shock; D1 = D1 receptor; FDA = U.S. Food and Drug Administration; HR = heart rate; NO = nitric oxide; PDE-3 = phosphodiesterase 3; SVR = systemic vascular resistance.

and enhance perfusion to vital organs.[51,52] Whenever a congestive phenotype exists, it should be addressed through intravenous loop diuretics, augmentation with thiazide diuretics, and renal replacement therapy for ultrafiltration if congestion is refractory to medical management. Failure to address congestion may lead to microcirculatory ischemia and damage to multiple organs, including the kidneys, liver, and gastrointestinal tract. In this regard, the transrenal perfusion pressure may serve as a useful conceptual model that represents the difference between the MAP and the central venous pressure. Hypoperfusion can be addressed by starting intravenous vasoactive medications, including inotropes, chronotropes, inopressors, inodilators, vasodilators, and vasopressors (Table 2).

These drugs are commonly used in clinical practice for the management of CS but some have been associated with increased adverse effects, including arrhythmias, and increased myocardial oxygen consumption.[51] Therefore, the writing committee advises that vasoactive medications be used in CS at the lowest possible dose to support adequate perfusion and for the shortest possible duration.

Inotropes enhance cardiac function by increasing the load-independent contractility of the myocardium. More specifically, cardiac calcitropes, including catecholamines and phosphodiesterase 3 inhibitors, increase the concentration of intracellular calcium to augment contractility. Notably, these agents exert their myocardial forces through direct action as opposed to secondary effects on chronotropy and vascular tone. Chronotropes, on the other hand, increase cardiac output by predominantly increasing the heart rate. Inopressors (eg, dopamine, norepinephrine, and epinephrine) increase cardiac output while increasing systemic vascular resistance, whereas inodilators (eg, milrinone or dobutamine) increase cardiac output and reduce afterload through systemic vasodilation. Vasodilators decrease preload and/or LV afterload through reduction in systemic vascular resistance, thereby potentially augmenting cardiac output. Pure vasopressors increase MAP by increasing systemic vascular resistance. The balance between alpha- and beta-adrenergic receptor activity determines the predominant effects of catecholamines, which run the gamut from pure inotropes to pure vasopressors (Table 2).

A Cochrane analysis found insufficient evidence to support the superiority of a particular inotrope or vasodilating agent in CS, especially with respect to a mortality benefit.[53] The Dobutamine Compared with Milrinone (DOREMI) trial evaluated dobutamine vs milrinone in 192 CS patients in a single quaternary care academic center, ranging from SCAI B through E and excluding patients with cardiac arrest, and found no difference with respect to the primary composite endpoint of in-hospital death from any cause, resuscitated cardiac arrest, receipt of a cardiac transplant or mechanical circulatory support,

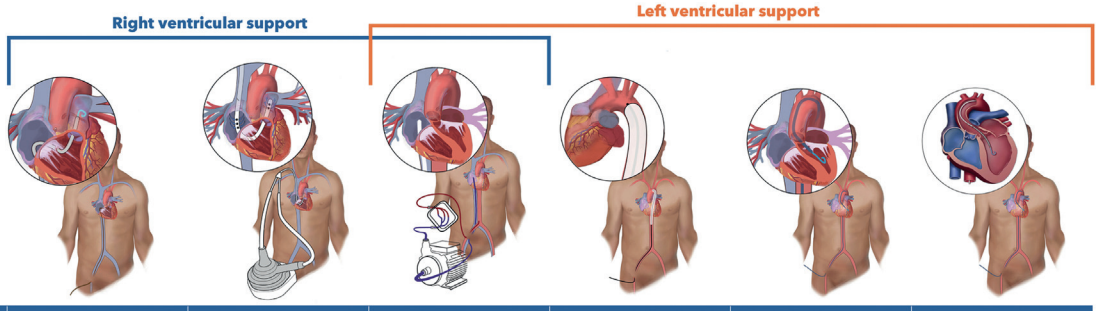
nonfatal myocardial infarction, transient ischemic attack or stroke diagnosed by a neurologist, or initiation of renal replacement therapy.[54] Given the heterogenous nature of CS and the various physiological derangements, several vasoactive medications may be attempted to normalize a CS patient's hemodynamic and/or metabolic profile. Inodilators or vasodilators may be considered in normotensive CS, especially in patients with increased systemic vascular resistance. Intravenous milrinone, given its relatively long half-life and renal excretion, should be judiciously used in patients with worsening renal function. Chronotropes might be trialed in bradycardia-induced CS. Since pure vasopressors (ie, phenylephrine) can cause reflex bradycardia and reduce cardiac output in CS, their use as a single first-line continuous intravenous medication is strongly discouraged. Although there is no clear consensus regarding the choice of first-line vasoactive agent, the writing committee agrees that norepinephrine is a reasonable first choice for most patients with CS who are hypotensive. Level 2 and 3 CS hospital centers should pursue consultation and consider potential transfer to a Level 1 CS hospital center when patients are refractory to the initial pharmacological therapy for consideration of tMCS support and/or advanced therapies.

### 4.6. tMCS

RCTs examining tMCS in CS have primarily been focused on STEMI-CS and have been limited by sample size, study design, patient selection, timing, and other key limitations. Few head-to-head randomized comparisons exist between various tMCS devices, and the potential therapeutic benefits must be weighed against bleeding, vascular, neurological, infectious, and other complications (Figure 6).[21]

Although tMCS is being increasingly utilized in the treatment of both AMI-CS and HF-CS,[55,56] routine use of tMCS in *all* CS patients is strongly discouraged. The Intraaortic Balloon Pump in Cardiogenic Shock (IABP-SHOCK) II trial was a multicenter, randomized, open-label

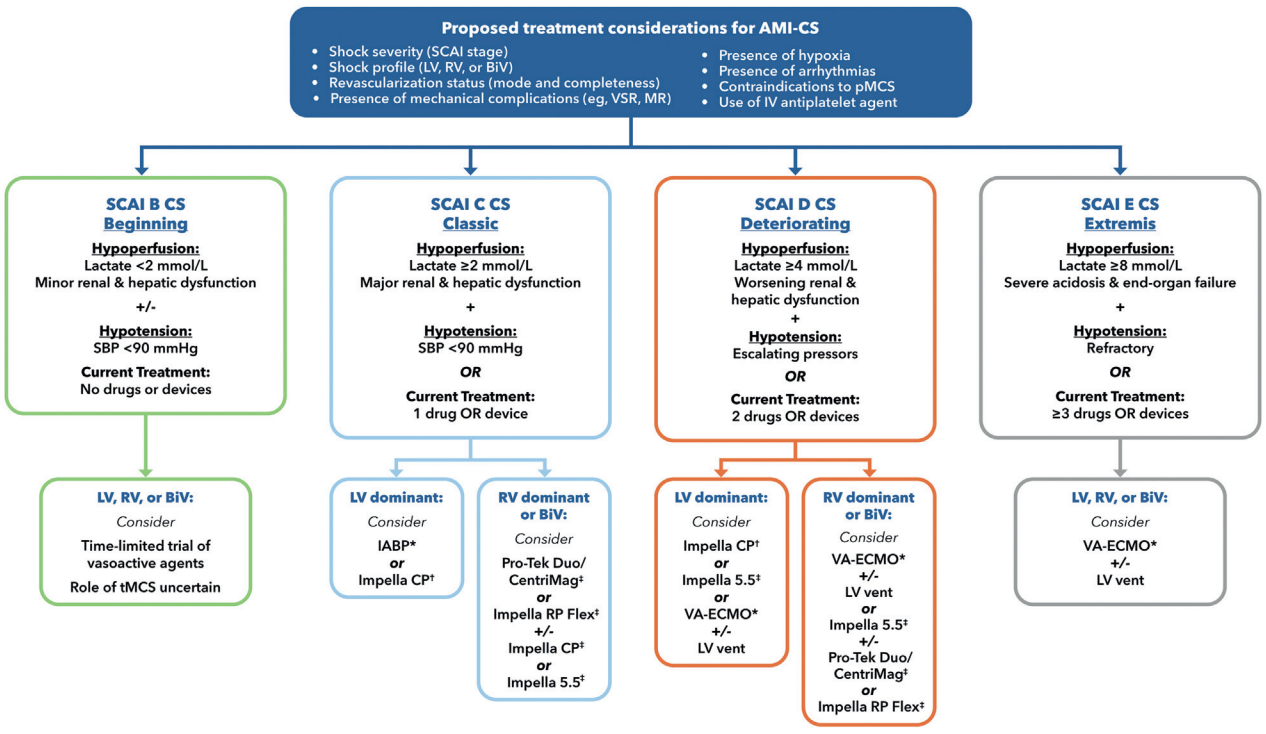
**FIGURE 6 Common tMCS Devices Used in CS**




| | Impella RP Flex | RA-PA pVAD | VA-ECMO | IABP | Impella CP | Impella 5.5 |
|---|---|---|---|---|---|---|
| Max flow | 3.0 - 4.0 L/min | 4.0 - 5.0 L/min | 5.0 - 7.0 L/min | 0.5 - 1.0 L/min | 3.0 - 4.3 L/min | 5.0 - 6.0 L/min |
| Max pump speed | 33,000 rpm | 7,500 rpm | 6,000 rpm | NA | 46,000 rpm | 33,000 rpm |
| Mechanism | Axial flow continuous pump (RA-to-PA) | Centrifugal flow continuous pump (RA-to-PA) | Centrifugal flow continuous pump (RA-to-AO) | Balloon inflation-deflation (AO) | Axial flow continuous pump (LV-to-AO) | Axial flow continuous pump (LV-to-AO) |
| Sheath size | 23 F venous peel-away | 29 or 31 F venous (inflow) | 15-24 F arterial<br>19-25 F venous | 7-8 F arterial | 14 F arterial peel-away | 23 F arterial peel-away |
| Typical insertion/ placement | Internal jugular vein | Internal jugular vein | Femoral vein (drain)<br>Femoral artery (return) | Femoral artery or Axillary artery | Femoral artery or Axillary artery | Axillary artery |
| Direct LV unloading | - | - | - | - | +++ | +++ |
| Direct RV unloading | + | + | + | - | - | - |
| Afterload | - | - | ↑↑↑ | ↓↓ | ↓ | ↓ |
| Coronary perfusion | - | - | ↑↑ | ↑↑ | ↑↑ | ↑↑ |

Adapted with permission from Tehrani BN, et al.[21] AO = aorta; CP = cardiac power; CS = cardiogenic shock; F = French; IABP = intra-aortic balloon pump; LV = left ventricular; NA = not applicable; PA = pulmonary artery; pVAD = percutaneous ventricular assist device; RA = right atrium; RA-PA = right atrium to pulmonary artery; RP = right percutaneous; rpm = revolutions per minute; RV = right ventricular; tMCS = temporary mechanical circulatory support; VA-ECMO = venoarterial extracorporeal membrane oxygenation.

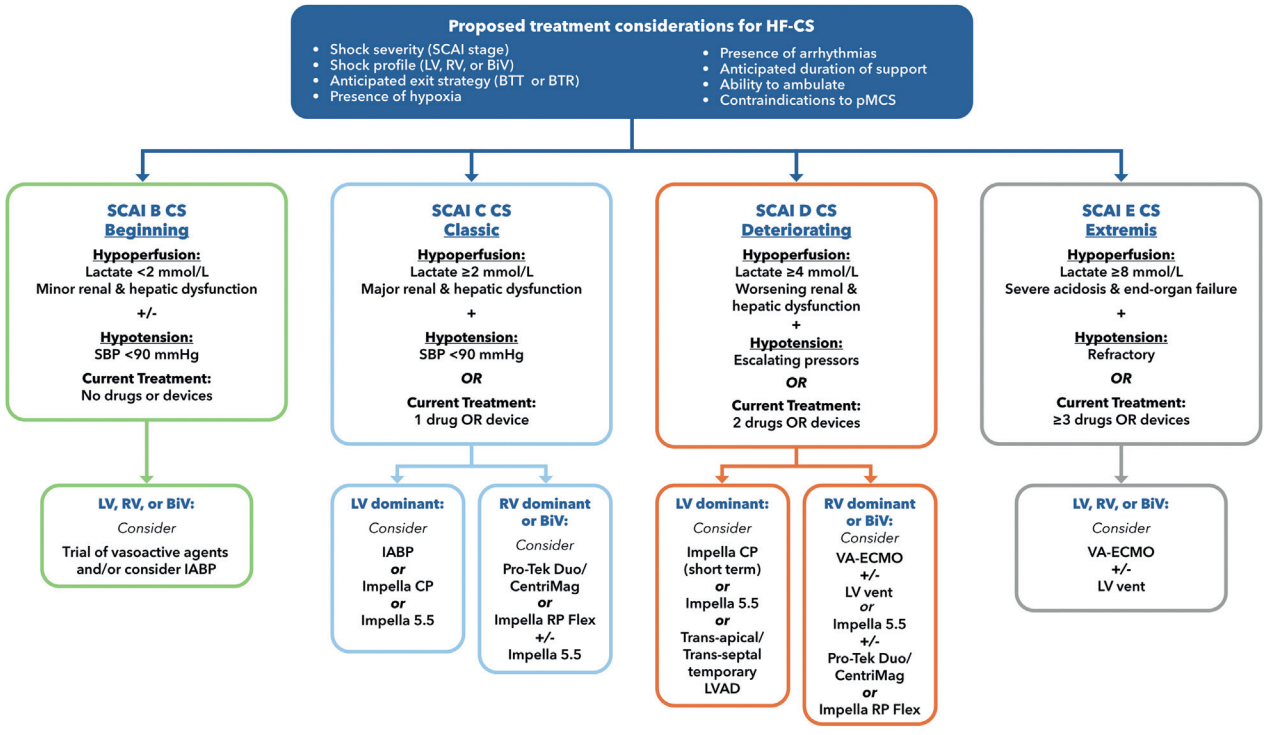
**FIGURE 7** **AMI-CS Management**




Adapted with permission from Mehta A, et al.[26] *Although tMCS have been studied in the IABP-Shock II and ECLS SHOCK trials, the results were neutral. Observational studies suggest some therapeutic benefit in this patient population. Routine use of these devices has not shown mortality benefit in RCTs. †Supported by evidence from DanGer (Danish-German) Shock trial. ‡Has not yet been studied in large RCTs. AMI = acute myocardial infarction; BiV = biventricular; CS = cardiogenic shock; ECLS-SHOCK = Extracorporeal Life Support in Cardiogenic Shock; IABP = intra-aortic balloon pump therapy; IABP-Shock = Intra-atoric Balloon Pump in Cardiogenic Shock; IV = intravenous; LV = left ventricle; MR = mitral regurgitation; pMCS = percutaneous mechanical circulatory support; RCT = randomized controlled trial; RV = right ventricle; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions; tMCS = temporary mechanical circulatory support; VA-ECMO = venoarterial extracorporeal membrane oxygenation; VSR = ventricular septal rupture.

trial of 600 patients with AMI-CS who were undergoing early revascularization with intra-aortic balloon pump vs control, and demonstrated no effect on 30-day all-cause mortality and at 6-year long-term follow-up.[57,58] The Extracorporeal Life Support in Infarct-Related Cardiogenic Shock (ECLS-SHOCK) trial was a multicenter, randomized, open-label trial of 420 patients with AMI-CS for whom early revascularization was planned, which showed that the 30-day all-cause mortality was not lower among patients who received early extracorporeal life support (ECLS group) with medical treatment vs usual medical treatment alone (control group).[59] Indeed, the DanGer Shock trial was the first trial that demonstrated that *early use* of a microaxial flow pump in *select* patients with STEMI-related shock, randomized within 24 hours of CS at experienced centers, improved 180-day survival as compared with standard of care, noting an absolute mortality reduction of 12.7%.[9a] The rigorous entry criteria for the DanGer Shock trial limit the evidence-base from the trial to a narrow cohort of patients with STEMI-CS.[60] An individual patient data meta-analysis of RCTs in CS with 6-month follow-up suggested that patients with STEMI-CS without risk of hypoxic brain injury had a reduction in mortality after tMCS use, inclusive of venoarterial extracorporeal membrane oxygenation (VA-ECMO).[61] The writing group believes escalation to tMCS with a microaxial flow-pump may be considered in appropriately selected STEMI-CS patients with LV-dominant shock who have evidence of clinical hypoperfusion and/or hemodynamic deterioration. Proposed standardized team-based algorithms, using the SCAI classification for risk stratification, with pharmacological and tMCS use in AMI-CS and HF-CS are shown in **Figures 7 and 8**, respectively, to provide a therapeutic framework

**FIGURE 8** **HF-CS Management**




Adapted with permission from Mehta A, et al.[26] Note: None of these tMCS devices have been studied in large RCTs in HF-CS. BiV = biventricular; BTR = bridge to recovery; BTT = bridge to transplantation; CS = cardiogenic shock; HF = heart failure; IABP = intra-aortic balloon pump therapy; LV = left ventricular; LVAD = left ventricular assist device; pMCS = percutaneous mechanical circulatory support; RCT = randomized controlled trial; RV = right ventricular; tMCS = temporary mechanical circulatory support; SBP = systolic blood pressure; SCAI = Society for Cardiovascular Angiography and Interventions; VA-ECMO = venoarterial extracorporeal membrane oxygenation.

for management.[26] The writing committee notes that Figures 7 and 8 incorporate several proposed therapies that have yet to be rigorously studied in well-designed, large, multicenter RCTs (especially in HF-CS), and thus further prospective, randomized investigation is needed.

From a mechanistic standpoint, the goal of tMCS is to promote ventricular unloading as well as restore systemic perfusion and is designed to bridge the CS patient to advanced therapies or facilitating myocardial recovery. In addition, the use of tMCS may allow de-escalation and weaning from pharmacological therapies that may exacerbate myocardial damage or ischemia if continued at high doses for prolonged duration.[3] The principles underlying addition or escalation of tMCS are as follows: using data if available, tMCS selection (including device capacity and risk of device-related complications) should be based on attaining the desired cardiac index to improve perfusion when pharmacological support alone is inadequate. If the CS patient is undersupported with respect to cardiac index on the present device, escalation of tMCS may be warranted. If the CS patient is not adequately unloaded on present support, the range of therapeutic options include increasing flow on current device, escalating support device (eg, Impella CP® to 5.5), addressing afterload (eg, LV venting for VA-ECMO), and mitigating congestion. Early palliative care consultation should be considered if not concurrently pursued at the time of tMCS placement, as some patients may not be candidates for durable LVAD, heart transplant, or myocardial recovery.[62]

Delays in initiation of tMCS in appropriate candidates may lead to worsening end-organ perfusion and hemometabolic derangements that ultimately culminate in multiorgan failure and death.[3,63] Thus, early CS recognition, prompt initiation of tailored and selective tMCS, and serial reassessment may improve outcomes.[27] Evidence of

pulmonary edema, persistent congestion, worsening perfusion, and/or multiorgan dysfunction should prompt team-based discussion for escalation of tMCS support. In particular, the first 24 hours appears to be critical in the management of CS, as most patients with CS change SCAI stages within the first 24 hours from CS diagnosis.[64,65] SCAI Stage B patients are at highest risk of worsening shock severity by 24 hours.[64,66] Further clinical trials are needed to determine which patients are most likely to benefit from tMCS devices, especially among HF-CS patients, which have not been well represented or studied in RCTs.

### 4.7. Critical Care Management

Key elements of the ongoing critical care of the patient with CS include: 1) serial reassessment of indicators of the adequacy of hemodynamic support; 2) treatment of reversible contributing causes of CS; 3) mitigation of complications of tMCS and advanced intensive care unit care; 4) management of end-organ injury resulting from CS; 5) de-escalation of invasive therapies as soon as possible; and 6) coordination of continued interdisciplinary decision making regarding the overall management and objectives of shock care, including myocardial recovery, advanced therapies (ie, durable LVAD, heart transplant), or palliative care/hospice.[67,68] Engaging patients, caregivers, and other family members throughout the patient's critical care journey is crucial.[69]

CS is dynamic and necessitates frequent structured reassessment to ensure that organ perfusion is being adequately supported and that therapies associated with risk are de-escalated whenever possible. The clinical exam, vital signs, laboratory measures of organ perfusion, imaging, and invasive hemodynamics (when available) comprise the key elements of this structured reassessment (Table 3). Some components are routinely measured continuously, whereas laboratory testing is periodically obtained (Table 3). Early after intensive care unit admission or when the severity is high, laboratory metrics of perfusion may be useful as frequently as every 2 to 4 hours and may be spaced to every 6 to 8 hours if the patient's condition stabilizes. These data enable reassessment of CS severity and determination of the need for escalation or de-escalation of hemodynamic support.

Complications of large-bore vascular access for mechanical circulatory support devices, including bleeding and limb ischemia, warrant vigilant monitoring and preventive interventions. In AMI-CS, the incidence of major bleeding may be as high as 60% and the risk of limb ischemia is 4-fold higher in patients requiring tMCS.[71,72] Limb ischemia is associated with a 2-fold higher risk of in-hospital death among CS patients,[72] yet diagnosis and management of acute limb ischemia in CS remains poorly understood. In addition to minimizing the duration of vascular access, helpful practices for reducing complications related to large-bore arterial devices are listed in Table 4.

If uncontrollable bleeding or limb ischemia occurs, prompt removal of the tMCS device is usually necessary. Removal of large-bore vascular access devices (≥12 French) in the catheterization laboratory or operating room should be considered whenever possible.[71]

Positive pressure ventilation is often necessary in the management of patients with CS and carries important cardiopulmonary interactions that may impact hemodynamic status of the patient.[73] Positive end-expiratory

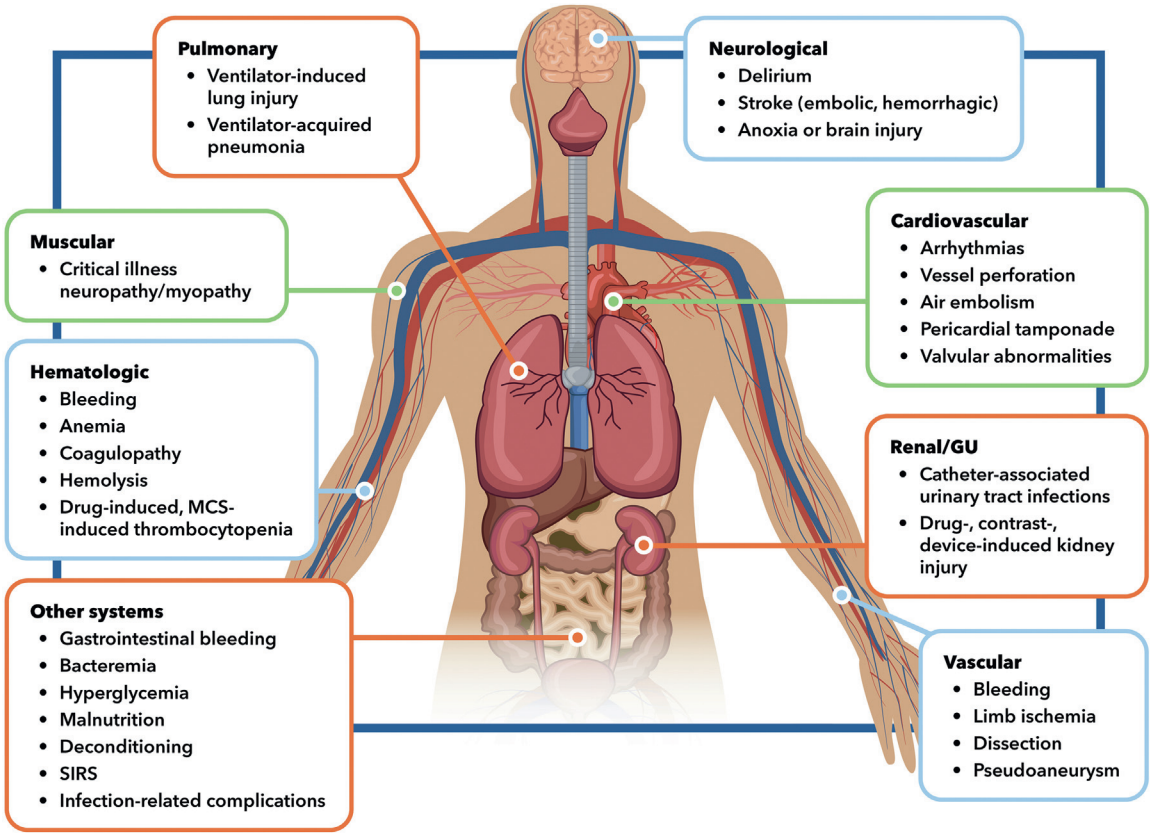
**TABLE 3 Structured Reassessment of Patients With CS in the Intensive Care Unit**

| Category | Measure | Frequency |
|---|---|---|
| **Clinical examination** | Mental status<br>Perfusion of extremities<br>Vascular access sites<br>Peripheral pulses (with arterial access in place) | Q6-8 h (Q1-2 h pulse exam with large-bore arterial vascular access in place) |
| **Vital signs** | Heart rate (and rhythm)<br>Mean arterial pressure<br>Arterial oxygen saturation | Continuous |
| **Physiological** | Urine output | Hourly |
| **Laboratory** | Serum creatinine<br>Serum bicarbonate<br>Arterial or venous pH<br>Central or mixed venous oxygen saturation<br>Lactate<br>Liver chemistries | Q2-8 h depending on phase of care (see text) |
| **Imaging** | Echocardiographic assessment of biventricular performance, intravascular volume, and MCS positioning (when applicable)<br>Chest radiography to aid in assessment of MCS/PAC/TVP/ETT positioning | Consider echo daily (may be limited point-of-care ultrasound)<br>Chest radiography may be performed as needed |
| **Invasive hemodynamics (if invasive monitoring in place)** | Central venous pressure<br>Pulmonary artery pressures<br>Pulmonary capillary wedge pressure<br>Estimated cardiac output | CVP and PA pressure are usually continuous<br>Estimated cardiac output may be continuous or Q2-8 h |
| **Interdisciplinary shock team** | Shock team assessment of clinical status | Daily (or more frequent when metrics indicate clinical worsening) |

The morbidity of CS is strongly associated with both cardiovascular and non-cardiovascular complications of critical care (Figure 9).[70-72]

CS = cardiogenic shock; CVP = central venous pressure; ETT = endotracheal tube; h = hour; MCS = mechanical circulatory support; PA = pulmonary artery; PAC = pulmonary artery catheter; pH = potential of hydrogen; Q = every; TVP = transvenous pacing lead.

**FIGURE 9** Systems Based Complications in CS




CS = cardiogenic shock; GU = genitourinary; MCS = mechanical circulatory support; SIRS = systemic inflammatory response syndrome.

pressure increases pulmonary vascular resistance, decreases RV and LV preload, decreases LV afterload, and reduces LV compliance through interventricular dependence. Therefore, the hemodynamic effects of positive end-expiratory pressure will vary depending upon how much preload dependence and LV contractility is present and whether RV failure is present (Figure 10).[73] Both positive pressure ventilation and positive end-expiratory pressure decrease LV diameter and increase transmural LV pressure. LV afterload decreases due to baroreceptor reflex response to aortic compression. These mechanisms augment LV stroke volume, thereby benefiting patients with LV failure with or without severe mitral regurgitation.[73]

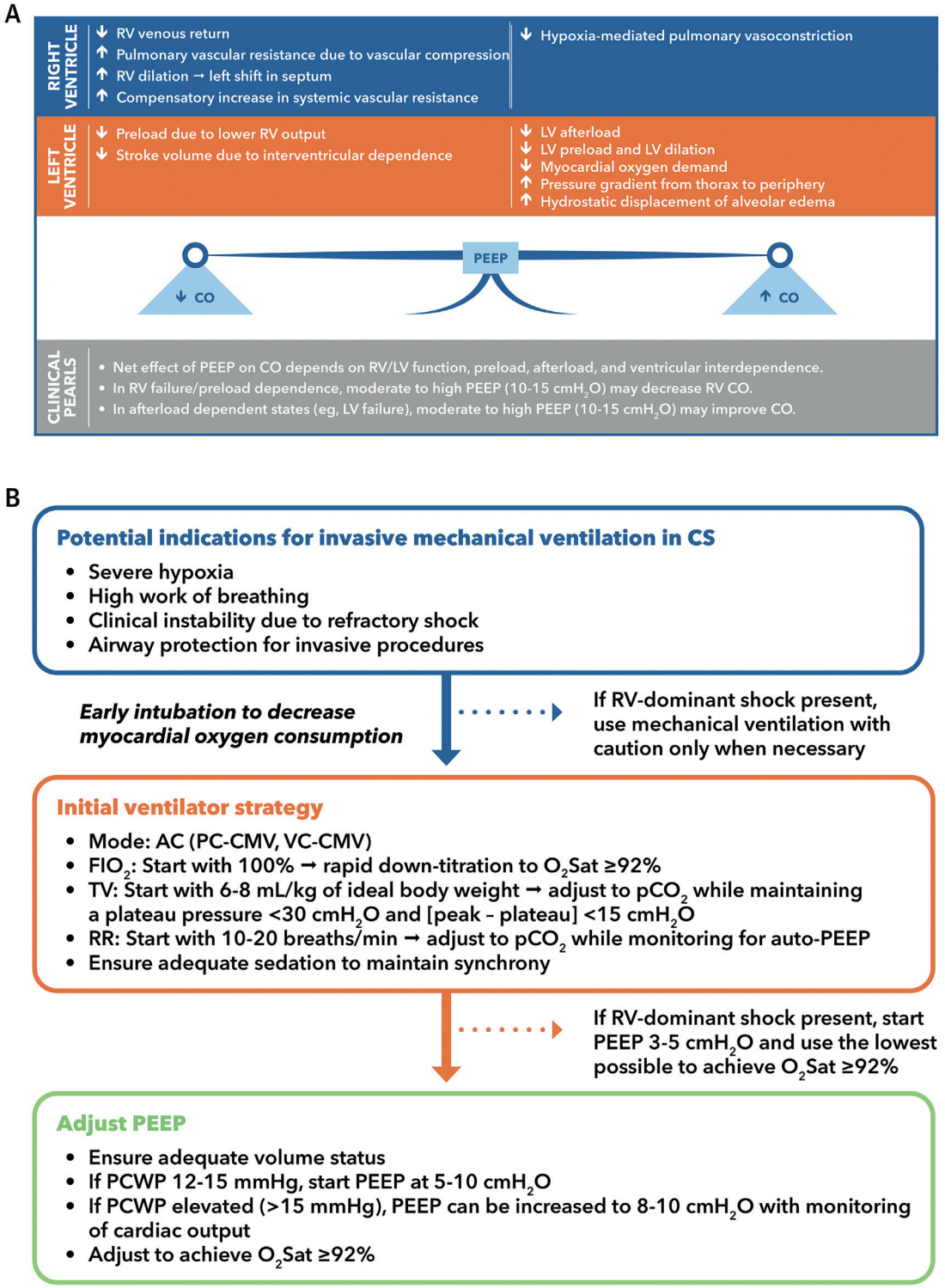
**TABLE 4** Measures to Reduce the Complications of Large-Bore Vascular Access in CS

| Measures to Reduce Complications |
|---|
| 1. Serial examination of vascular access sites and limbs for bleeding and ischemia |
| 2. Avoidance of excessive anticoagulation |
| 3. Examination of vascular access sites after mobilizing the patient for turns and off-unit studies |
| 4. Attention to the angulation of vascular access catheter insertion and securement to avoid tenting of the arteriotomy site |

CS = cardiogenic shock.

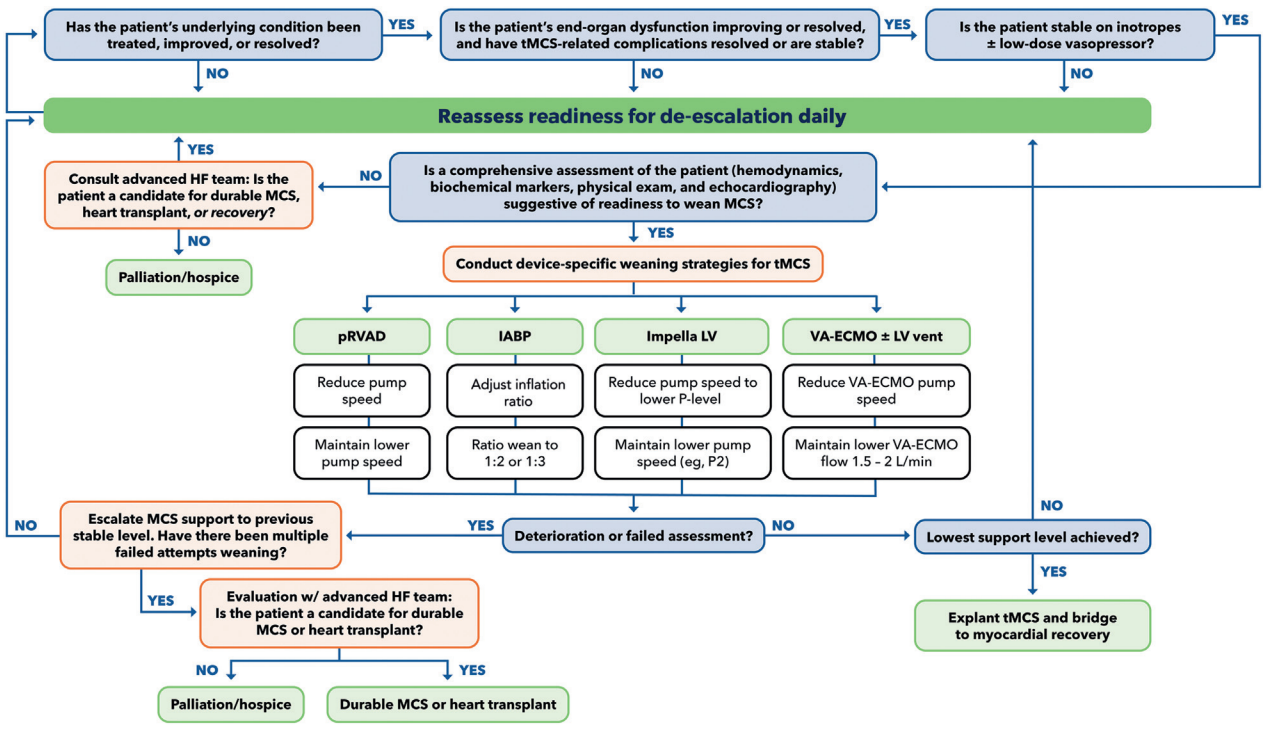
Decision-making regarding the weaning of tMCS is a central element of the interdisciplinary critical care management of patients with CS. Rigorous evidence to guide weaning of mechanical circulatory support is lacking. A daily assessment of readiness to wean should include evaluation of hemodynamic stability, consideration of the current total need for pharmacological vasoactive support, volume status, and whether the underlying cause of CS has been corrected or improved.[27,74] A rational approach to weaning includes a stepwise decrease in flow from the device, the pace of which will be dictated by the nature of the initial cardiovascular insult and its reversibility. Before

**FIGURE 10** Cardiopulmonary Interactions of PEEP (A) and Strategy for Mechanical Ventilation (B) in Patients With CS


A
RIGHT VENTRICLE
↓ RV venous return
↑ Pulmonary vascular resistance due to vascular compression
↑ RV dilation → left shift in septum
↑ Compensatory increase in systemic vascular resistance
↓ Hypoxia-mediated pulmonary vasoconstriction
LEFT VENTRICLE
↓ Preload due to lower RV output
↓ Stroke volume due to interventricular dependence
↓ LV afterload
↓ LV preload and LV dilation
↓ Myocardial oxygen demand
↑ Pressure gradient from thorax to periphery
↑ Hydrostatic displacement of alveolar edema
PEEP
↓ CO
↑ CO
CLINICAL PEARLS
• Net effect of PEEP on CO depends on RV/LV function, preload, afterload, and ventricular interdependence.
• In RV failure/preload dependence, moderate to high PEEP (10-15 cmH2O) may decrease RV CO.
• In afterload dependent states (eg, LV failure), moderate to high PEEP (10-15 cmH2O) may improve CO.
B
Potential indications for invasive mechanical ventilation in CS
• Severe hypoxia
• High work of breathing
• Clinical instability due to refractory shock
• Airway protection for invasive procedures
Early intubation to decrease myocardial oxygen consumption
If RV-dominant shock present, use mechanical ventilation with caution only when necessary
Initial ventilator strategy
• Mode: AC (PC-CMV, VC-CMV)
• FIO2: Start with 100% → rapid down-titration to O2Sat ≥92%
• TV: Start with 6-8 mL/kg of ideal body weight → adjust to pCO2 while maintaining a plateau pressure <30 cmH2O and [peak - plateau] <15 cmH2O
• RR: Start with 10-20 breaths/min → adjust to pCO2 while monitoring for auto-PEEP
• Ensure adequate sedation to maintain synchrony
If RV-dominant shock present, start PEEP 3-5 cmH2O and use the lowest possible to achieve O2Sat ≥92%
Adjust PEEP
• Ensure adequate volume status
• If PCWP 12-15 mmHg, start PEEP at 5-10 cmH2O
• If PCWP elevated (>15 mmHg), PEEP can be increased to 8-10 cmH2O with monitoring of cardiac output
• Adjust to achieve O2Sat ≥92%

Adapted with permission from Alviar CL, et al.[73] (A) Cardiopulmonary interactions of PEEP in patients with CS. (B) Strategy for mechanical ventilation in patients with CS. AC = assist control; $cmH_2O$ = centimeter of water; CO = cardiac output; CS = cardiogenic shock; $FiO_2$ = fraction of inspired oxygen; LV = left ventricular; $O_2Sat$ = oxygen saturation; PC-CMV = pressure-controlled continuous mechanical ventilation; $pCO_2$ = partial pressure of carbon dioxide; PCWP = pulmonary capillary wedge pressure; PEEP = positive end-expiratory pressure; RR = respiratory rate; RV = right ventricular; TV = tidal volume; VC-CMV = volume-controlled continuous mechanical ventilation.

**FIGURE 11** Weaning tMCS


Has the patient's underlying condition been treated, improved, or resolved?
YES
Is the patient's end-organ dysfunction improving or resolved, and have tMCS-related complications resolved or are stable?
YES
Is the patient stable on inotropes ± low-dose vasopressor?
YES
NO
NO
NO
Reassess readiness for de-escalation daily
YES
Consult advanced HF team: Is the patient a candidate for durable MCS, heart transplant, or recovery?
NO
Palliation/hospice
NO
Is a comprehensive assessment of the patient (hemodynamics, biochemical markers, physical exam, and echocardiography) suggestive of readiness to wean MCS?
YES
Conduct device-specific weaning strategies for tMCS
pRVAD
IABP
Impella LV
VA-ECMO ± LV vent
Reduce pump speed
Adjust inflation ratio
Reduce pump speed to lower P-level
Reduce VA-ECMO pump speed
Maintain lower pump speed
Ratio wean to 1:2 or 1:3
Maintain lower pump speed (eg, P2)
Maintain lower VA-ECMO flow 1.5 - 2 L/min
NO
Escalate MCS support to previous stable level. Have there been multiple failed attempts weaning?
YES
Deterioration or failed assessment?
NO
Lowest support level achieved?
NO
YES
YES
Evaluation w/ advanced HF team: Is the patient a candidate for durable MCS or heart transplant?
Explant tMCS and bridge to myocardial recovery
NO
YES
Palliation/hospice
Durable MCS or heart transplant

Adapted with permission from Randhawa VK, et al.[74] *No single metric should be used in isolation to determine weaning suitability. CS = cardiogenic shock; HF = heart failure; IABP = intra-aortic balloon pump; LV = left ventricle; MCS = mechanical circulatory support; P = pump speed setting; pRVAD = percutaneous right ventricular assist device; tMCS = temporary mechanical circulatory support; VA-ECMO = veno-arterial extracorporeal membrane oxygenation.

weaning, it is reasonable to consult an advanced HF/transplant cardiologist to determine whether the patient is a suitable candidate for myocardial recovery versus durable LVAD, or heart transplant. The success of weaning is evaluated at each step by an integrated assessment of clinical exam, metrics of end-organ perfusion, imaging, and invasive hemodynamic data, when available, with a standard weaning interval aiming for 0.5- 1L/min decrease in support (eg, 2 performance levels with Impella) every 2 to 4 hours (**Figure 11**).[27,74] CS patients, especially those due to acute-on-chronic HF-CS, may require a bridge from tMCS with inotropes, and the latter may require a prolonged wean in certain instances.

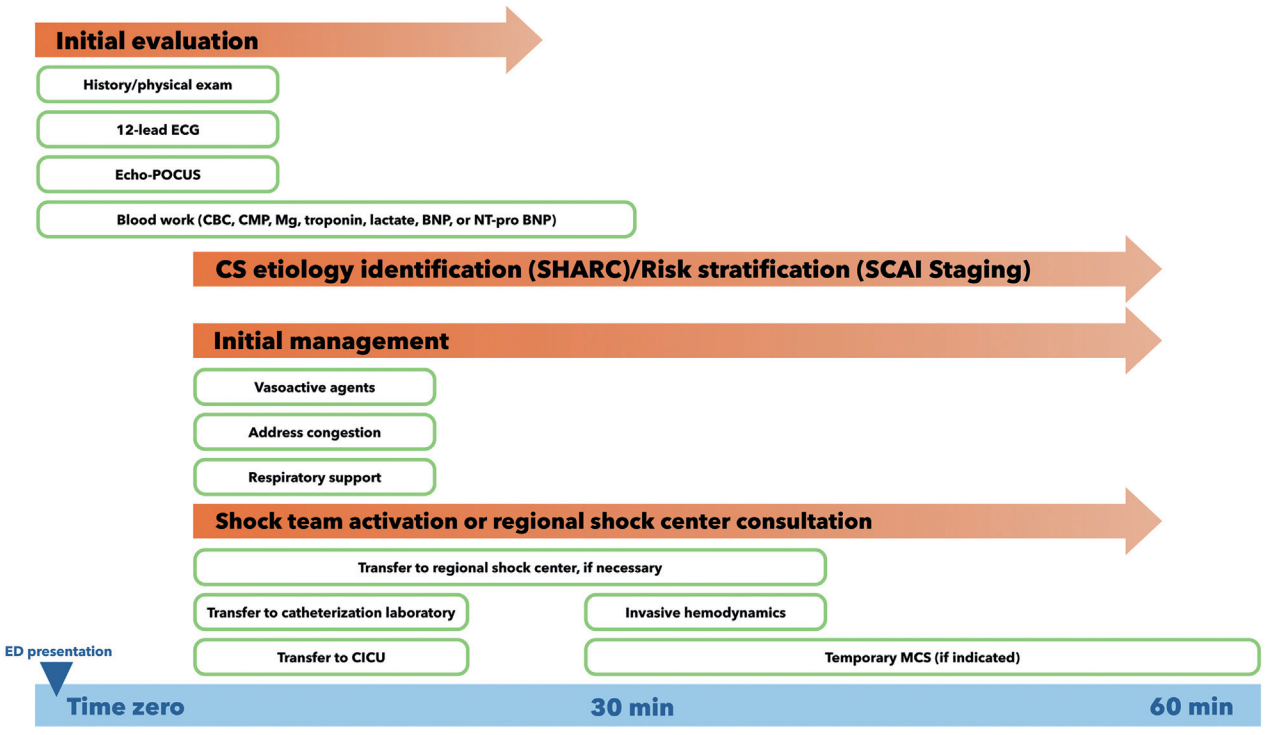
The optimal treatment of CS represents a multiphase, team-based approach tailored to the patient's hemodynamic and metabolic profile. In this manner, the Recognize/Rescue - Optimize - Stabilize - De-Escalation/Exit framework may serve as a useful heuristic.[75]

The first phase of therapy is to recognize CS and restore adequate tissue perfusion ("recognize/rescue" [**Figure 12**]).[75] Following this phase, the goal is to tailor the pharmacological support to achieve hemodynamic stability with MAP >60 to 65 mm Hg considered a reasonable hemodynamic target ("optimization").[75] The "stabilization" phase is characterized by recovery of end-organ function and mitigation of extra-cardiac derangements. The "de-escalation/exit" phase enables assessment of myocardial recovery, and if not, transition to advanced therapies, such as durable LVAD or orthotopic heart transplant for appropriate candidates.[75]

### 4.8. Conclusion

CS remains a hemodynamically complex, multifactorial syndrome with high morbidity and mortality. A high index of suspicion is necessary to promote early recognition, confirm the diagnosis and initiate prompt pharmacological and/or tMCS therapy based on risk stratification. Invasive hemodynamic monitoring may be useful in guiding therapy selection and escalation of support. Serial reassessment, particularly within the first 24 hours, is advised to ensure hemodynamic stability, restore tissue perfusion, and mitigate end-organ damage. In this manner, a standardized team-based approach has been associated with improved outcomes and can facilitate the patient's transition to myocardial recovery, advanced therapies, or palliative care/hospice, as appropriate.[18]

**FIGURE 12** Golden Hour of Acute Shock




Adapted with permission from Polyzogopoulou E, et al.[18] Sources for this figure adaptation: Waksman R, et al.[1] and Naidu SS, et al.[2] Note: this figure is intended to be used primarily in patients who experience AMI-CS and acute HF-CS. AMI-CS = acute myocardial infarction-cardiogenic shock; BNP = brain-type natriuretic peptide; CBC = complete blood count; CICU = cardiac intensive care unit; CMP = comprehensive metabolic panel; CS = cardiogenic shock; ECG = electrocardiogram; ED = emergency department; HF-CS = heart failure-cardiogenic shock; MCS = mechanical circulatory support; Mg = magnesium; NT-proBNP = N-terminal pro b-type natriuretic peptide; POCUS = point-of-care ultrasound; SCAI = Society for Cardiovascular Angiography and Interventions; SHARC = Shock Academic Research Consortium.

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**KEY WORDS** acute myocardial infarction, cardiogenic shock, concise clinical guidance, critical care, heart failure, hemodynamics, shock

## APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT)—2025 CONCISE CLINICAL GUIDANCE: AN ACC EXPERT CONSENSUS STATEMENT ON THE EVALUATION AND MANAGEMENT OF CARDIOGENIC SHOCK

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ACC = American College of Cardiology; ECP = expandable cardiac power; SHIELD = Surgical site Hospital acquired Infection prEvention with Local D-plex; TIMI = Thrombolysis In Myocardial Infarction.

## APPENDIX 2. PEER REVIEWER RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (COMPREHENSIVE)—2025 CONCISE CLINICAL GUIDANCE: AN ACC EXPERT CONSENSUS STATEMENT ON THE EVALUATION AND MANAGEMENT OF CARDIOGENIC SHOCK

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†No financial benefit.

‡Relationship with this company is limited to enrolling patients in clinical trials. This disclosure was entered under the Clinical Trial Enroller category in the ACC's disclosure system. To appear in this category, the author acknowledges that there is no direct or institutional relationship with the trial sponsor as defined in the ACC/AHA Disclosure Policy for Writing Committees.

ACC = American College of Cardiology; AHA = American Heart Association; ARIES = Aspirin-Free Regimen in Patients with HeartMate 3 LVAD; ASAIO = American Society for Artificial Internal Organs; ASPC = American Society of Preventive Cardiology; DSMB = Data Safety Monitoring Board; JACC = *Journal of the American College of Cardiology*; JHLT = *Journal of Heart and Lung Transplantation*; MCS = Mechanical Circulatory Support; NIH = National Institutes of Health; PCORI = Patient-Centered Outcomes Research Institute; SSOC = Solution Set Oversight Committee; STS = Society of Thoracic Surgeons; VCSQI = Virginia Cardiac Services Quality Initiative; VHAC = Virginia Heart Attack Coalition.

## APPENDIX 3. ABBREVIATIONS

ACC = American College of Cardiology
AMI = acute myocardial infarction
AMI-CS = acute myocardial infarction-cardiogenic shock
CS = cardiogenic shock
HF = heart failure
HF-CS = heart failure-cardiogenic shock
LVAD = left ventricular assist device
LV = left ventricular
MAP = mean arterial pressure
MI = myocardial infarction
RCT = randomized controlled trial
RV = right ventricular
SCAI = Society for Cardiovascular Angiography and Interventions
STEMI = ST-segment elevation myocardial infarction
STEMI-CS = ST-segment elevation myocardial infarction-cardiogenic shock
tMCS = temporary mechanical circulatory support